

=> s atherosclerosis or arteriosclerosis  
L2 226621 ATHEROSCLEROSIS OR ARTERIOSCLEROSIS

=> s tibolone or 5630-53-5/rn  
'RN' IS NOT A VALID FIELD CODE  
'RN' IS NOT A VALID FIELD CODE  
'RN' IS NOT A VALID FIELD CODE  
L3 1428 TIBOLONE OR 5630-53-5/RN

=> s 12 and 13  
L4 49 L2 AND L3

=> dup rem 14  
PROCESSING COMPLETED FOR L4  
L5 35 DUP REM L4 (14 DUPLICATES REMOVED)

=> s 15 and py<1997  
2 FILES SEARCHED...  
L6 6 L5 AND PY<1997

=> d 16 1-6 ab bib kwic

L6 ANSWER 1 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
AB This review summarizes recent data on the effects of endogenous and exogenous androgens, estrogens and progesterone on serum lipoproteins levels and composition in humans. Sex steroid hormones modulate serum lipoprotein metabolic mechanisms and influence **atherosclerosis** and coronary heart disease. In general, androgens lower HDL and raise LDL levels and Lp(a) thus promoting the atherogenic process. As it is true with estrogens, the lipoprotein effects of androgens are more pronounced with oral than with parenteral administration. Millions of women use oral contraception and postmenopausal women use more and more some form of hormone replacement therapy. The HDL-raising effect of estrogen replacement seems to be mediated by an increase in apoprotein AI production and not by a decrease in the clearance rate. Estrogens lower LDL levels by accelerating the rate of LDL catabolism which is due to an increase in the number of hepatic LDL receptors. They also improve endothelium-dependent vasodilatation which might be mediated by an antioxidant action of estrogens. These facts could explain well known cardioprotective effects of estrogens. Androgen progestins, especially older such as norgestrel, lower HDL and raise LDL thus diminishing or eliminating the benefits of estrogens on cardiovascular system while

newer

progestins have a lesser effect on circulating lipoproteins.  
AN 96168299 EMBASE  
DN 1996168299  
TI [The effects of androgens and other sex steroid hormones on serum lipoproteins].  
UCINCI ANDROGENA I DRUGIH SPOLNIH HORMONA NA SERUMSKE LIPOPROTEINE.  
AU Reiner Z.  
CS Klinika za Unutarnje Bolesti, Klinicki Bolnicki Centar 'Rebro',  
Kispaticeva 12,Zagreb, Croatia  
SO Lijecnicki Vjesnik, (1996) 118/SUPPL. 1 (33-37).  
ISSN: 0024-3477 CODEN: LIVJA5  
CY Croatia  
DT Journal; Conference Article  
FS 003 Endocrinology  
010 Obstetrics and Gynecology  
029 Clinical Biochemistry  
030 Pharmacology

037 Drug Literature Index

LA Serbo-Croatian

SL English; Serbo-Croatian

SO Lijecnicki Vjesnik, (1996) 118/SUPPL. 1 (33-37).  
ISSN: 0024-3477 CODEN: LIVJA5

AB . . . and progesterone on serum lipoproteins levels and composition in humans. Sex steroid hormones modulate serum lipoprotein metabolic mechanisms and influence **atherosclerosis** and coronary heart disease. In general, androgens lower HDL and raise LDL levels and Lp(a) thus promoting the atherogenic process.. . .

CT Medical Descriptors:  
\*lipoprotein blood level  
**atherosclerosis**  
conference paper  
female  
hormone substitution  
human  
ischemic heart disease  
lipoprotein metabolism  
oral drug administration  
\*androgen: PD, pharmacology  
\*androgen: EC, endogenous compound  
\*estrogen: EC, endogenous compound  
\*estrogen: PD, pharmacology  
\*lipoprotein: EC, endogenous. . . compound  
\*sex hormone: PD, pharmacology  
\*sex hormone: EC, endogenous compound  
danazol: PD, pharmacology  
medroxyprogesterone acetate: PD, pharmacology  
norethisterone: PD, pharmacology  
norgestrel: PD, pharmacology  
stanazolol: PD, pharmacology  
testosterone: PD, pharmacology  
**tibolone: PD, pharmacology**

RN (progesterone) 57-83-0; (danazol) 17230-88-5; (medroxyprogesterone acetate) 71-58-9; (norethisterone) 68-22-4; (norgestrel) 6533-00-2; (stanazolol) 10418-03-8, 302-96-5; (testosterone) 58-22-0; (**tibolone**) 5630-53-5

L6 ANSWER 2 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Menopause is the physiologic state that is a consequence of the cessation of ovarian function. A large number of vasomotor, psychological and gynecological symptoms have been associated with menopause. Hormonal replacement therapy is effective in treating these conditions. The use of estrogens and progestins including dosages, routes of administration and their advantages and disadvantages are reviewed in this article. In addition, hormonal replacement therapy may reduce the risk of **atherosclerosis** and prevent the osteoporosis of climateric women. Hormonal therapy is associated with side effects but they do not contraindicate its use.

AN 95116094 EMBASE

DN 1995116094

TI [Hormonal replacement therapy in the climaterium].  
TERAPIA DE SUSTITUCION HORMONAL EN EL CLIMATERIO.

AU Canto De Cetina T.E.

CS Depto. de Biol. de la Reproduccion, Ctro. de Invest. Reg. Dr. H. Noguchi, Universidad Autonoma, Calle 59 No. 490, Merida, Yucatan, Mexico

SO Revista de Investigacion Clinica, (1995) 47/1 (49-61).  
ISSN: 0034-8376 CODEN: RICLAG

CY Mexico

DT Journal; General Review  
FS 003 Endocrinology  
010 Obstetrics and Gynecology  
020 Gerontology and Geriatrics  
030 Pharmacology  
037 Drug Literature Index  
LA Spanish  
SL Spanish; English  
SO Revista de Investigacion Clinica, (1995) 47/1 (49-61).  
ISSN: 0034-8376 CODEN: RICLAG  
AB . . . and their advantages and disadvantages are reviewed in this article. In addition, hormonal replacement therapy may reduce the risk of **atherosclerosis** and prevent the osteoporosis of climateric women. Hormonal therapy is associated with side effects but they do not contraindicate its. . .  
CT Medical Descriptors:  
\*climacterium  
\*menopause  
drug . . .  
acetate: AE, adverse drug reaction  
mestranol: AE, adverse drug reaction  
norethisterone: AE, adverse drug reaction  
norgestrel: AE, adverse drug reaction  
progesterone: AE, adverse drug reaction  
**tibolone: AE, adverse drug reaction**  
RN (chlormadinone) 1961-77-9; (estradiol) 50-28-2; (estrone) 53-16-7;  
(ethinylestradiol) 57-63-6; (medroxyprogesterone acetate) 71-58-9;  
(mestranol) 72-33-3; (norethisterone) 68-22-4; (norgestrel) 6533-00-2;  
(progesterone) 57-83-0; (**tibolone**) 5630-53-5  
L6 ANSWER 3 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
AB Lipoprotein (a) has been implicated with an increased risk of **atherosclerosis** and cardiovascular disease. Recently, considerable progress has been made toward understanding the importance of genetics in the regulation of plasma levels of lipoprotein (a). However, the issue as to whether lipoprotein (a) levels should be treated is still debated and furthermore the possibilities to influence lipoprotein (a) levels remain limited. The potential clinical importance of Lipoprotein (a) has stimulated interest in the dietary and pharmacologic agents that affect this lipoprotein. At present, only a few of the existing therapeutic tools, such as nicotinic acid and estrogens, have been found to consistently affect lipoprotein (a).  
AN 95071854 EMBASE  
DN 1995071854  
TI Diet and drug therapy for lipoprotein (a).  
AU Berglund L.  
CS Div of Preventive Medicine Nutrition, Dept Medicine, College of Physicians, Surgeons of Columbia University, 630 West 108th Street, New York, NY 10032, United States  
SO Current Opinion in Lipidology, (1995) 6/1 (48-56).  
ISSN: 0957-9672 CODEN: COPLEU  
CY United Kingdom  
DT Journal; General Review  
FS 037 Drug Literature Index  
038 Adverse Reactions Titles  
LA English  
SL English  
SO Current Opinion in Lipidology, (1995) 6/1 (48-56).  
ISSN: 0957-9672 CODEN: COPLEU  
AB Lipoprotein (a) has been implicated with an increased risk of

**atherosclerosis** and cardiovascular disease. Recently, considerable progress has been made toward understanding the importance of genetics in the regulation of plasma. . .

CT Medical Descriptors:

- \*cardiovascular disease: DT, drug therapy
- \*cardiovascular disease: PC, prevention
- \*cardiovascular disease: TH, therapy
- \*diet therapy
- apheresis
- atherosclerosis: DT, drug therapy**
- atherosclerosis: PC, prevention**
- atherosclerosis: TH, therapy**
- cholesterol diet
- clinical trial
- double blind procedure
- eskimo
- fat intake
- female
- human
- hypercholesterolemia
- hyperlipoproteinemia: CO, complication
- kidney disease
- lipoprotein blood level
- lipoprotein metabolism
- low calory diet
- male
- medical genetics
- mouse
- non insulin dependent diabetes mellitus
- nonhuman
- postmenopause
- primate
- priority. . .
- pharmacology
- nicotinic acid: AE, adverse drug reaction
- octreotide
- pravastatin: DT, drug therapy
- pravastatin: CB, drug combination
- probucol: DT, drug therapy
- stanazolol: DT, drug therapy
- tamoxifen: PD, pharmacology
- thyroid hormone
- tibolone: DT, drug therapy**
- very low density lipoprotein: EC, endogenous compound

RN. . . 1404-04-2, 1405-10-3, 8026-22-0; (niceritrol) 5868-05-3; (nicotinic acid) 54-86-4, 59-67-6; (octreotide) 83150-76-9; (pravastatin) 81131-74-0;

(probucol) 23288-49-5; (stanazolol) 10418-03-8, 302-96-5; (tamoxifen) 10540-29-1; (**tibolone**) 5630-53-5

L6 ANSWER 4 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 92315534 EMBASE

DN 1992315534

TI The role of ovarian and testicular steroids in metabolism of lipids and in atherogenesis.

AU Marek J.

CS III Interni Klinika, I Lekarska Fakulta, Univerzita Karlova, U nemocnice 1,128 21 Praha, Czechoslovakia

SO Vnitřní Lékařství, (1992) 38/9 (913-920).

ISSN: 0042-773X CODEN: VNLEAH

CY Czechoslovakia

DT Journal; Article

FS 003 Endocrinology

005 General Pathology and Pathological Anatomy

030 Pharmacology

037 Drug Literature Index

038 Adverse Reactions Titles

018 Cardiovascular Diseases and Cardiovascular Surgery

LA Czech

SL English; Czech

SO Vnitřní Lekarství, (1992) 38/9 (913-920).

ISSN: 0042-773X CODEN: VNLEAH

CT Medical Descriptors:

- \*atherosclerosis: SI, side effect
- \*atherosclerosis: PC, prevention
- \*atherosclerosis: DT, drug therapy
- \*hypercholesterolemia: SI, side effect
- \*hypercholesterolemia: DT, drug therapy
- \*hypercholesterolemia: PC, prevention
- \*lipid metabolism
- \*lipoprotein metabolism
- article
- cardiovascular disease
- drug effect
- female
- heart protection
- hormone substitution
- human
- male
- menopause
- \*estrogen: AE, adverse drug reaction
- \*estrogen: PD, pharmacology
- \*estrogen: DT, drug therapy
- \*gestagen: AE, adverse drug reaction
- \*tamoxifen: DT, drug therapy
- \*testosterone derivative: DT, drug therapy
- \*tibolone: DT, drug therapy
- allylestrenol
- conjugated estrogen
- estradiol
- estriol
- estrofem
- levonorgestrel
- lynestrenol
- medroxyprogesterone acetate
- mestranol
- methyltestosterone: DT, drug therapy
- norethisterone
- norgestrel
- tamoxifen citrate

RN (tamoxifen) 10540-29-1; (**tibolone**) 5630-53-5; (allylestrenol) 432-60-0; (estradiol) 50-28-2; (estriol) 50-27-1; (estrofem) 65296-29-9; (levonorgestrel) 797-63-7; (lynestrenol) 52-76-6; (medroxyprogesterone acetate) 71-58-9; (mestranol) 72-33-3; (methyltestosterone) 58-18-4; .

L6 ANSWER 5 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Org OD 14 is a synthetic steroid which in animal bioassays displays oestrogenic as well as very weak androgenic-anabolic properties. Earlier

studies have shown that it alleviates oestrogen-deficiency symptoms and retards osteoporosis. OD 14 can be administered continuously with little effect on the endometrium. The aim of this study was to evaluate the effect of OD 14 on apolipoprotein A1 (Apo-A1), the major protein constituent of the high-density lipoprotein (HDL) fraction, as compared with that of oestradiol valerate (E2V) and a placebo. Twenty-two women, who had been oophorectomized when undergoing surgical treatment for stage IB or IIA cervical carcinoma, were given OD 14 2.5 mg/day, a placebo, and E2V 2 mg/day for a period of 6 wk in each case using a double-blind, cross-over method. Serum Apo-A1 was determined by electro-immunoassay after each treatment period. There was a marked decrease in Apo-A1 after OD 14 as compared with the levels seen after the placebo and E2V. This decrease is interpreted as evidence of a strong androgenic influence by

OD

14. In epidemiological studies low levels of Apo-A1 have been associated with a higher incidence of **atherosclerosis** and cardiovascular disease. Long-term treatment with OD 14 might therefore be hazardous in this respect.

AN 85088669 EMBASE

DN 1985088669

TI Apolipoprotein A1 levels in oophorectomized women treated with Org OD 14, oestradiol valerate and a placebo.

AU Crona N.; Silfverstolpe G.; Enk L.; Samsioe G.

CS Department of Obstetrics and Gynecology, Sahlgrenska Hospital, Goteborg, Sweden

SO Maturitas, (1984) 6/4 (335-339).

CODEN: MATUDK

CY Netherlands

DT Journal

FS 037 Drug Literature Index

010 Obstetrics and Gynecology

030 Pharmacology

003 Endocrinology

LA English

SO Maturitas, (1984) 6/4 (335-339).

CODEN: MATUDK

AB . . . androgenic influence by OD 14. In epidemiological studies low levels of Apo-A1 have been associated with a higher incidence of **atherosclerosis** and cardiovascular disease. Long-term treatment with OD 14 might therefore be hazardous in this respect.

CT Medical Descriptors:

**\*atherosclerosis**

\*cardiovascular disease

\*lipid blood level

\*drug therapy

\*uterine cervix carcinoma

ovariectomy

priority journal

therapy

human

blood and hemopoietic system

female genital system

clinical article

endocrine system

cardiovascular system

\*apolipoprotein a1

\*estradiol valerate

\*placebo

**\*tibolone**

RN (estradiol valerate) 979-32-8; (**tibolone**) 5630-53-5

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS

AB The effects of daily 2.5 mg/day doses of 7.alpha.,17.alpha.-hydroxy-7-methyl-19-norpregn-5(10)-en-20-yn-3-one (Org OD 14) on lipid metab., with particular ref. to high-d. lipoprotein (HDL)-related variables, were assessed in 14 healthy post-menopausal women after 12 and 36 mo of treatment. There were significant redns. in the following variables

after

both treatment periods: total phospholipids, total triglycerides, HDL-phospholipids and apolipoprotein A1. No changes were obsd. in total cholesterol or low-d. lipoprotein (LDL) cholesterol over the entire treatment period. A temporary decrease was obsd. in HDL-cholesterol

after

12 mo, with a return to pretreatment values after 36 mo of treatment.

The

findings of this study clearly show that Org OD 14 has no adverse effects on the atherogenic variables, viz. LDL-cholesterol and triglycerides. Indeed, since the latter were lowered, its action is in fact beneficial. Moreover, its effect on HDL-cholesterol, the antiatherogenic variable, is only temporary. Although the compn. of HDL changes during Org OD 14 treatment (esp. as regards its cholesterol content), there is no evidence that reverse cholesterol transport is impaired.

AN 1990:509446 CAPLUS

DN 113:109446

TI Long-term effects of Org OD 14 on lipid metabolism in post-menopausal women

AU Kloosterboer, H. J.; Benedek-Jaszmann, L. J.; Kicovic, P. M.

CS Organon Sci. Dev. Group, Oss, 5340 BH, Neth.

SO Maturitas (1990), 12(1), 37-42

CODEN: MATUDK; ISSN: 0378-5122

DT Journal

LA English

SO Maturitas (1990), 12(1), 37-42

CODEN: MATUDK; ISSN: 0378-5122

IT **Atherosclerosis**

(Org OD 14 effect on lipid metab. in postmenopausal women in relation to)

IT **5630-53-5**, Org OD 14

RL: BIOL (Biological study)

(lipid metab. response to long-term administration of, in postmenopausal women)

=>